WHAT IS CLAIMED IS:

1. A method for identifying a compound that modulates cell cycle arrest, the method comprising the steps of:

(i) contacting a cell comprising a target polypeptide or fragment thereof or inactive variant thereof, selected from the group consisting of flap structure specific endonuclease 1 (FEN1), protein kinase C ζ (PKC-ζ), phospholipase C-β1 (PLC-β1), protein tyrosine kinase 2 (FAK), protein tyrosine kinase 2b (FAK2), casein kinase 2 (CK2), cMET tyrosine kinase (cMET), REV1 dCMP transferase (REV1), apurinic/apyrimidinic nuclease 1 (APE1), cyclin dependent kinase 3 (CDK3), PIM1 kinase (PIM1), cell division cycle 7 kinase (CDC7L1), cyclin dependent kinase 7 (CDK7), cytokine inducible kinase (CNK), potentially prenylated protein tyrosine phosphatase (PRL-3), serine threonine kinase 2 (STK2) or (NEK4), cyclin dependent serine threonine kinase (NKIAMRE), or histone acetylase (HBO1), or fragment thereof with the compound, the target polypeptide encoded by the complement of a nucleic acid that hybridizes under stringent conditions to a nucleic acid encoding a polypeptide having an amino acid sequence selected from the group consisting of SEQ ID NO:14, 2, 4, 6, 8, 10, 12, 16, 18, 20, 22, 24, 26, 28, 30, 32, 34, and 36; and

(ii) determining the chemical or phenotypic effect of the compound upon the cell comprising the target polypeptide or fragment thereof or inactive variant thereof, thereby identifying a compound that modulates cell cycle arrest.

2. The method of claim 1, wherein the chemical or phenotypic effect is determined by measuring enzymatic activity selected from the group consisting of nuclease activity, kinase activity, lipase activity, transferase activity, phosphatase activity, and acetylase activity.

3. The method of claim 1, wherein the chemical or phenotypic effect is determined by measuring cellular proliferation.

4. The method of claim 3, wherein the cellular proliferation is measured by assaying fluorescent marker level or DNA synthesis.

5. The method of claim 4, wherein DNA synthesis is measured by ^{3}H thymidine incorporation, BrdU incorporation, or Hoescht staining.

6. The method of claim 4, wherein the fluorescent marker is selected from the group consisting of a cell tracker dye or green fluorescent protein.

7. The method of claim 1, wherein modulation is activation of cell cycle arrest.

8. The method of claim 1, wherein modulation is activation of cancer cell cycle arrest.

9. The method of claim 1, wherein the host cell is a cancer cell.

10. The method of claim 9, wherein the cancer cell is a breast, prostate, colon, or lung cancer cell.

11. The method of claim 9, wherein the cancer cell is a transformed cell line.

12. The method of claim 11, wherein the transformed cell line is A549, PC3, H1299, MDA-MB-231, MCF7, or HeLa.

13. The method of claim 9, wherein the cancer cell is p53 null or mutant.

14. The method of claim 9, wherein the cancer cell is p53 wild-type.

15. The method of claim 1, wherein the polypeptide is recombinant.

16. The method of claim 1, wherein the polypeptide is encoded by a nucleic acid comprising a sequence of SEQ ID NO:13, 1, 3, 5, 7, 9, 11, 15, 17, 19, 21, 23, 25, 27, 29, 31, 33, or 35.

17 . The method of claim 1, wherein the compound is an antibody.

18 . The method of claim 1, wherein the compound is a small organic molecule.

19 . The method of claim 1, wherein the compound is an antisense molecule.

20. The method of claim 1, wherein the compound is a peptide.

21. The method of claim 20, wherein the peptide is circular.

22. The method of claim 1, wherein the compound is an siRNA molecule.

23. A method for identifying a compound that modulates cell cycle arrest, the method comprising the steps of:

(i) contacting a cell comprising a target polypeptide or fragment thereof or inactive variant thereof, selected from the group consisting of flap structure specific endonuclease 1 (FEN1), protein kinase C ζ (PKC-ζ), phospholipase C-β1 (PLC-β1), protein tyrosine kinase 2 (FAK), protein tyrosine kinase 2b (FAK2), casein kinase 2 (CK2), cMET tyrosine kinase (cMET), REV1 dCMP transferase (REV1), apurinic/apyrimidinic nuclease 1 (APE1), cyclin dependent kinase 3 (CDK3), PIM1 kinase (PIM1), cell division cycle 7 kinase (CDC7L1), cyclin dependent kinase 7 (CDK7), cytokine inducible kinase (CNK), potentially prenylated protein tyrosine phosphatase (PRL-3), serine threonine kinase 2 (STK2) or (NEK4), cyclin dependent serine threonine kinase (NKIAMRE), or histone acetylase (HBO1), or fragment thereof with the compound, the target polypeptide encoded by the complement of a nucleic acid that hybridizes under stringent conditions to a nucleic acid encoding a polypeptide having an amino acid sequence selected from the group consisting of SEQ ID NO:14, 2, 4, 6, 8, 10, 12, 16, 18, 20, 22, 24, 26, 28, 30, 32, 34, and 36; and

(ii) determining the physical effect of the compound upon the target polypeptide or fragment thereof or inactive variant thereof; and

(iii) determining the chemical or phenotypic effect of the compound upon a cell comprising the target polypeptide or or fragment thereof or inactive variant thereof, thereby identifying a compound that modulates cell cycle arrest.

24. A method of modulating cell cycle arrest in a subject, the method comprising the step of administering to the subject a therapeutically effective amount of a compound identified using the method of claim 1.

25. The method of claim 24, wherein the subject is a human.

26. The method of claim 25, wherein the subject has cancer.

27. The method of claim 24, wherein the compound is a small organic molecule.

28. The method of claim 24, wherein the compound is an antisense molecule.

29. The method of claim 24, wherein the compound is an antibody.

30. The method of claim 24, wherein the compound is a peptide.

31. The method of claim 30, wherein the peptide is circular.

32. The method of claim 24, wherein the compound is an siRNA molecule.

33. The method of claim 24, wherein the compound inhibits cancer cell proliferation.

34. A method of modulating cell cycle arrests in a subject, the method comprising the step of administering to the subject a therapeutically effective amount of a target polypeptide or fragment thereof or inactive variant thereof, selected from the group consisting of flap structure specific endonuclease 1 (FEN1), protein kinase C ζ (PKC-ζ), phospholipase C-β1 (PLC-β1), protein tyrosine kinase 2 (FAK), protein tyrosine kinase 2b (FAK2), casein kinase 2 (CK2), cMET tyrosine kinase (cMET), REV1 dCMP transferase (REV1), apurinic/apyrimidinic nuclease 1 (APE1), cyclin dependent kinase 3 (CDK3), PIM1 kinase (PIM1), cell division cycle 7 kinase (CDC7L1), cyclin dependent kinase 7 (CDK7), cytokine inducible kinase (CNK), potentially prenylated protein tyrosine phosphatase (PRL-3), serine threonine kinase 2 (STK2) or (NEK4), cyclin dependent serine threonine kinase (NKIAMRE), or histone acetylase (HBO1), or fragment thereof with the compound, the target polypeptide encoded by the complement of a nucleic acid that hybridizes under stringent conditions to a nucleic acid encoding a polypeptide having an amino acid sequence selected from the group consisting of SEQ ID NO:14, 2, 4, 6, 8, 10, 12, 16, 18, 20, 22, 24, 26, 28, 30, 32, 34, and 36.

35. A method of modulating cell cycle arrest in a subject, the method comprising the step of administering to the subject a therapeutically effective amount of a nucleic acid encoding a target polypeptide or fragment thereof or inactive variant thereof, selected from the group consisting of flap structure specific endonuclease 1 (FEN1), protein kinase C ζ (PKC-ζ), phospholipase C-β1 (PLC-β1), protein tyrosine kinase 2 (FAK), protein tyrosine kinase 2b (FAK2), casein kinase 2 (CK2), cMET tyrosine kinase (cMET), REV1 dCMP transferase (REV1), apurinic/apyrimidinic nuclease 1 (APE1), cyclin dependent kinase 3 (CDK3), PIM1 kinase (PIM1), cell division cycle 7 kinase (CDC7L1), cyclin dependent kinase 7 (CDK7), cytokine inducible kinase (CNK), potentially prenylated protein tyrosine phosphatase (PRL-3), serine threonine kinase 2 (STK2) or (NEK4), cyclin dependent serine threonine kinase (NKIAMRE), or histone acetylase (HBO1), or fragment thereof with the compound, the target polypeptide encoded by the complement of a nucleic acid that hybridizes under stringent conditions to a nucleic acid encoding a polypeptide having an amino acid sequence selected from the group consisting of SEQ ID NO:14, 2, 4, 6, 8, 10, 12, 16, 18, 20, 22, 24, 26, 28, 30, 32, 34, and 36.

36. A CK2-specific siRNA molecule comprising the sequence AACATTGAATTAGATCCACGT, wherein the siRNA molecule is from 21 to 30 nucleotide base pairs in length.

37. The CK2-specific siRNA molecule of claim 36 consisting of the sequence AACATTGAATTAGATCCACGT and its complement as active portion.

38. A method of inhibiting expression of a CK2 gene in a cell, the method comprising contacting the cell with a CK2-specific siRNA molecule comprising the sequence AACATTGAATTAGATCCACGT, wherein the siRNA molecule is from 21 to 30 nucleotide base pairs in length.

39. A PIM1-specific siRNA molecule comprising the sequence AAAACTCCGAGTGAACTGGTC, wherein the siRNA molecule is from 21 to 30 nucleotide base pairs in length.

40. The PIM1-specific siRNA molecule of claim 39 consisting of the sequence AAAACTCCGAGTGAACTGGTC and its complement as active portion.

41. A method of inhibiting expression of a PIM1 gene in a cell, the method comprising contacting the cell with a PIM1-specific siRNA molecule comprising the sequence AAAACTCCGAGTGAACTGGTC, wherein the siRNA molecule is from 21 to 30 nucleotide base pairs in length.

42. An Hbo1-specific siRNA molecule comprising the sequence AACTGAGCAAGTGGTTGATTT, wherein the siRNA molecule is from 21 to 30 nucleotide base pairs in length.

43. The Hbo1-specific siRNA molecule of claim 42 consisting of the sequence AACTGAGCAAGTGGTTGATTT and its complement as active portion.

44. A method of inhibiting expression of an Hbo1 gene in a cell, the method comprising contacting the cell with an Hbo1-specific siRNA molecule comprising the sequence AACTGAGCAAGTGGTTGATTT, wherein the siRNA molecule is from 21 to 30 nucleotide base pairs in length.